**UNITED STATES DISTRICT COURT**
**SOUTHERN DISTRICT OF NEW YORK**

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| **NEW YORK STATE RESTAURANT ASSOCIATION,**<br><br>**Plaintiff,**<br><br>**- against -**<br><br>**NEW YORK CITY BOARD OF HEALTH, NEW YORK CITY DEPARTMENT OF HEALTH AND MENTAL HYGIENE, and Thomas R. Frieden,**<br>**In His Official Capacity as Commissioner of the New York City Department of Health and Mental Hygiene,**<br><br>**Defendants.** | **No. 2008 Civ. ____**<br><br>**:DECLARATION OF DAVID B. ALLISON, Ph.D** |

David B. Allison, Ph.D declares under penalty of perjury as follows:

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## A. Background and Qualifications

I am a Professor in the Departments of Biostatistics, Nutrition Sciences, Medicine, and Genetics, and Director of the National Institutes of Health-funded Clinical Nutrition Research Center at the University of Alabama at Birmingham. I am also a Senior Scientist at the Center for

Outcomes and Effectiveness Research and Education at the University of Alabama at Birmingham. In my current position, and in previous positions, I have conducted extensive research on the causes, consequences, and treatment of obesity; on the development and evaluation of statistical methods; and on study design. I have played a major role in the design, analysis, and interpretation of studies involving weight loss interventions. I received a Ph.D. in Psychology from Hofstra University in 1990 and am a licensed psychologist in the State of NY.

I am currently President-Elect of *NAASO: The Obesity Society*, the largest and most prestigious academic society for the study of obesity in North America, and will become President in October 2008. I have authored, in whole or in part, over 300 articles appearing in peer-reviewed journals. Many of these articles address nutrition, obesity, weight-loss, study design, and statistical analysis. I have also edited, authored, or co-authored multiple text books and chapters in text books on the topics of nutrition, obesity, and weight-loss. I have served on the editorial boards of the journals Obesity, Obesity Reviews, Nutrition Today, International Journal of Obesity, International Journal of Eating Disorders and Evidence-Based Preventive Medicine.

I previously served as an Associate Research Scientist at the NIH-funded New York Obesity Research Center at Saint Luke's/Roosevelt Hospital Center and as Associate Professor of Medical Psychology in Psychiatry at Columbia University in New York City. At the New York Obesity Research Center, I was primarily involved with designing and conducting original research in human obesity and related areas, collaborating with other investigators on their projects, particularly with respect to data analysis, and training students, interns, and post-doctoral fellows with respect to obesity research projects.

I have periodically served as a consultant to the Federal Trade Commission (FTC), the Food and Drug Administration (FDA), the United States Postal Inspection Service, and the United States Department of Justice on the validity of weight-loss product claims. In that capacity, I have reviewed numerous clinical research studies in order to determine whether efficacy claims are supported by the research. In 2006, I hosted, organized and spoke at a 2-day conference on *Design, Analysis and Interpretation of Randomized Clinical Trials in Obesity*. This conference was funded by the NIH through a grant on which I was the principal investigator.

I have been elected as a fellow of the American College of Nutrition and of the American Statistical Association, and am a member of the New York Academy of Sciences.

My experience, training, and research expertise qualify me to offer informed opinions on the evidential basis or lack thereof for claims that the New York City regulation requiring chain restaurants to post the calorie content of standardized menu items in a specified manner will help reverse an epidemic of obesity among New York City residents.

My curriculum vita is attached as Exhibit A and includes a more detailed description of my training and experience, and a listing of my publications.

**B. <u>Materials Reviewed and Relied Upon.</u>**

1. I reviewed the original NYC Board of Health's *Notice of Adoption of a Resolution To Repeal and Reenact §81.50 of the New York City Health Code*, the new "*Department of Health and Mental Hygiene, Board of Health, Notice of Adoption of a Resolution To Repeal and Reenact §81.50 of The New York City Health Code*" and the studies cited in those Notices in support of the contention that the regulation will impact

the amount of calories New Yorkers consume and will thereby help prevent obesity. I also reviewed the July 5, 2007 Declaration of Thomas R. Frieden, MD, MPH and many of the articles cited by Frieden in support of his supposition that the previously proposed Regulation 81.50 would have had an effect in reducing obesity. I have also reviewed the amicus briefs filed by the Center for Science in the Public Interest and by the City of San Francisco Attorney's Office regarding Regulation 81.50, and many articles cited therein. I have also conducted a review of the scientific articles bearing on the topic and have reviewed those that appeared most relevant and cite key articles in the references to this document.

## C. Opinions to be Offered and Propositions to Be Addressed

The issues under consideration are complex and there is a multiplicity of ideas involved. Hence to avoid any misconstrual, I wish to begin by clarifying not only what I see as the fundamental issues and what I will offer opinions on, but also what I will *not* address and which opinions I am *not* offering.

### C.1. Issues on Which I Will Not Opine.

My goal is to provide an assessment of whether there is scientific evidence showing that the revised Regulation 81.50 (hereafter *R81.50*) will achieve the objective of reducing obesity levels among NYC residents and/or people that dine in NYC. This is an empirical question that can be addressed via a consideration of scientific evidence. In contrast, whether R81.50 is 'good,' legally defensible, fair, adverse to legitimate economic interests, or 'should' be adopted are questions of a legal and social nature and I will ***not*** be addressing those questions. I will attempt to provide scientific information that can be used as inputs in the social and legal decision making process.

However, I am ***not*** offering the opinion that **(1)** R81.50 should not be adopted; nor am I offering the opinion that **(2)** R81.50 should be adopted.

**C.2. Issues on Which I will Opine.**

I believe that the propositions or issues that are germane to the scientific basis or lack thereof for adopting R81.50 can be grouped into three categories: **(1)** Those that support concern about obesity and suggest that as a society we should be seeking ways to reduce obesity and/or the deleterious effects it causes. I address these propositions in section E of this report; **(2)** Those that support the conjecture that providing calorie information at the point of purchase in restaurants (especially fast food restaurants) might be beneficial in reducing obesity levels. I address these propositions in section F of this report; and **(3)** Those that, if true, would directly indicate that implementation of R81.50 would be beneficial in reducing obesity levels. I address these propositions in section G of this report.

## D. Standards of Evidence

Before proceeding to an evaluation of relevant evidence, it is important to clarify the standard of evidence I will refer to herein. I refer to scientific evidence and by evidence I mean facts that could potentially lead to a reasonable *conclusion* that causation exists as opposed to facts that could lead one to *conjecture* or *hypothesize* about putative causation. Clearly, this standard requires that we seek evidence generated using procedures generally accepted in the scientific community as capable of supporting valid conclusions as to causation.

When evaluating outcome claims about interventions in humans, there are clear standards accepted by the biomedical research community. These standards are articulated in a number of publications relating to testing effects of interventions in

general (e.g., Meinert et al., 1986), interventions for weight control (Allison et al., 1997; Committee for Proprietary Medicinal Products, 1997; FDA, 1996; Gadbury et al., 2003; Anderson et al.,1998), and in the legal context specifically (Green et al., 2000). Although there may be nuances to studying effects with one type of intervention (product, program, substance) as compared to another, the same general principles of sound experimental design, statistical analysis, and interpretation apply.

A key point to consider when reviewing purportedly scientific opinions is the joint concept of objectivity and observability in the scientific process. The process leading to conclusions should be an objective one that is articulatable and observable and, therefore, can be checked and reproduced by other scientists as well as evaluated for validity. To illustrate this point, consider the fact that many scientific journals, including the prestigious *Proceedings of the National Academy of Sciences*, ask their peer-reviewers to answer the following question when evaluating manuscripts that have been submitted for publication "Are the procedures described sufficiently well that the work can be repeated?" Simply stating that one has reviewed evidence and come to a conclusion without describing what the *objective* criteria for reaching such a conclusion are and showing that those criteria were met does not make something a valid scientific opinion.

**Types of Information that are Generally Accepted by the Scientific Community as Sufficient Evidence that an Intervention Aimed at Preventing Obesity Is Effective.**

With respect to assessing any possible effects of an intervention to reduce obesity, including both putative beneficial and deleterious effects, the ideal source of evidence would be one or more large randomized controlled trials (RCTs) of the intervention, in a sample from the population of interest. Importantly, these RCTS must

be well designed, well executed, and well analyzed. An RCT is a study in which humans are assigned to two or more conditions via a random process. Those conditions should ideally be identical in all respects except for the independent variable (i.e., 'treatment') under study. The group getting the treatment or intervention is generally referred to as the treatment or experimental group and the group that does not get the treatment in question is called the control group. After some appropriate period, all subjects in each of the two groups are then measured on the outcome (i.e., dependent variable) and compared with respect to the outcome using an appropriate statistical test.

However, for practical or ethical reasons, it is often impossible to conduct an RCT to address a particular question. In such situations, an observational (epidemiologic) study might be the best alternative, and evidence from such studies, though imperfect, would be the key evidence sought. In an observational study one studies people who were exposed (through their own choice or other circumstances) to the intervention of interest and compares them to people who were not so exposed. Observational epidemiologic studies cannot unequivocally demonstrate causality, but can document associations[1]. Associations observed in observational epidemiologic studies are more compelling with respect to possible inference to causation when the study is well designed, conducted, and analyzed. Again, one or more studies finding supportive results are not sufficient to support a claim if they are only a subset of a larger number

[1] In the field of statistics, two variables (e.g., a putative cause and a putative outcome) are said to be associated if and only if they are not independent. Two variables are independent if and only if the conditional distribution of one variable is identical for all levels of the other variable. Association differs from and does not necessarily imply causation. Causation means that one of the variables influences the other.

of studies some of which obtain contradictory evidence and the weight of evidence is not supportive.

Critically, because randomization is not used in observational epidemiologic studies, potential confounding factors must be delineated, carefully measured, and carefully controlled for in the statistical analysis. Unlike RCTs which can control for both known and unknown confounding factors, observational epidemiologic studies can only control for confounders[2] to the extent that the confounding variables can be effectively measured and for which the functional form of the relation between the potential confounding variable and the outcome can be effectively modeled in the statistical analysis.

In evaluating the body of evidence bearing on whether a treatment or intervention is effective, it has become standard in 'evidence-based' medicine and public health to rank studies according to objective criteria that include consideration of whether the data comes from RCTs or observational studies, the consistency of the data, and other aspects of study design and methodology. One authoritative set of criteria is that used by the National Heart, Lung and Blood Institute in its "Clinical Guidelines on the Identification, Evaluation and Treatment of Obesity - The Evidence

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[2] Confounding and confounders have been defined as follows:

> ***confounder or confounding factor:*** *A cause of something being studied ( i.e a disease) whose effect on that disease is mixed up with the effect (or non-effect) of another factor because the "confounder" is associated with that other factor. See "confounding".*
>
> ***confounding:*** *Epidemiologists use the term when the impact of two risk factors are associated with the same exposure and must be disentangled. Heavy alcohol consumption and smoking are both known to cause esophageal cancer. If people who drink also tend to smoke, then the effect of drinking will confound the effect of smoking and vice versa. Therefore one must correct for this confounding in the way the data are analyzed. Sometimes the non-effect of a factor which conveys no risk at all is confounded with the true effect of another factor. ...*
>
> **See**: http://www.dhs.ca.gov/ehib/emf/RiskEvaluation/Appendix8.pdf

Report" (2000). Those criteria rate evidence from a series of well-designed RCTs that provide a consistent pattern of findings in the population for which a recommendation is made as "Category A" evidence. Where there are only a few RCTs or findings are less than consistent, the evidence is ranked "Category B". Evidence from observational studies is rated "Category C". Evidence based only on expert clinical judgment is rated lowest, as "Category D". These or similar categories have been applied in assessing the evidence that public health interventions aimed at improving diet or reducing caloric consumption among the general public have been shown to be efficacious (Faith et al, 2007; see Seymour et al, 2004).

**E. Remarks on Propositions Supporting Concern about Obesity.**

The City, Dr. Frieden, and the amicus brief (hereafter simply *amicus*) previously filed by the City of San Francisco Attorney's Office all offer a number of propositions, and evidence in support of those propositions that, if true, justify substantial concern about obesity and suggest that as a society we should be seeking solutions. These propositions can be summarized in the following points.

*Proposition E1*. During the last several decades, the prevalence of obesity increased substantially in the world, the nation, and in NYC and is now sufficiently high as to represent a major public health concern.

*Proposition E2*. Obesity has many deleterious effects for the individual including reduced longevity, reduced quality of life, and reduced health and may also be costly to society.

I believe that *Propositions E1 and E2* are true and have indeed published many papers providing the evidence that would support them (see CV attached). Moreover, I believe that the evidence supporting these points has been well documented by the

City, Dr. Frieden, and the amicus, and therefore I will not dwell on them further. I also agree with the apparent conclusion of the City, Dr. Frieden, and the amicus that accepting *Propositions E1 and E2* justifies substantial concern about obesity and suggests that we should be seeking solutions.

**F. Remarks on Propositions Supporting Conjecture that Providing Calorie Information at The Point of Purchase In Restaurants (Especially Fast Food Restaurants) Might Be Beneficial in Reducing Obesity Levels.**

The City, Dr. Frieden, and the amicus advance several other propositions that, if true, I believe support the conjecture that providing calorie information at the point of purchase in restaurants (especially fast food restaurants) might be beneficial in reducing obesity levels. These propositions may be stated as follows.

*Proposition F1*. Obesity largely results from extended periods of positive energy balance (i.e., periods where energy intake exceeds energy expenditure).

*Proposition F2*. On average, Americans eat more food in restaurants now (in both absolute calories and as a proportion of total caloric intake) than Americans did in the past.

*Proposition F3*. Many of the foods served in restaurants are quite energy dense[3] and quite high in absolute calories.

*Proposition F4*. On average, Americans substantially underestimate the calories of food served in restaurants, largely as a function of large portion sizes.

*Proposition F5*. Restaurants and restaurant food are contributing to the obesity epidemic in some manner that is above and beyond the contribution offered by any source of food energy.

[3] Energy density refers to the energy ('calories') in foods divided by the weight (grams) of those foods.

I accept all of Propositions F1 to F3 and think that they are sufficiently well documented (including in materials presented by the City, Dr. Frieden, and the amicus) to not merit further discussion. With respect to Proposition F4, I do believe that there are reasons to question it. People may not report their true beliefs about the calorie content of foods they typically eat accurately in part because doing so may help to preserve their self-image and public personae as 'reasonable' eaters. We have previously shown evidence that would suggest this may be the case (Muhlheim et al., 1998). Nevertheless, I suspect that even were this putative bias taken into account, people would still be shown to underestimate the caloric content of large restaurant (and also large non-restaurant) meals and food items. Thus, for the purposes of further discussion, I will accept the validity of Proposition F4.

In contrast, the support for Proposition F5 is more open to question. Clearly, acceptance of proposition F1 implies that any source of food energy consumed by humans is contributing to the obesity epidemic. This is as true for fresh fruits, vegetables and lean broiled fish as it is for French fries, ice cream, and refined sugar. But this statement is more tautological than informative. Acceptance of Proposition F1 does not *ipso facto* imply that restaurants and restaurant food are contributing to the obesity epidemic in some manner that is *above and beyond* the contribution offered by any source of food energy.

Though acceptance of Propositions F1 to F4 make it plausible that Proposition F5 is true, there are several reasons to retain some skepticism about the truth (or lack thereof) of Proposition F5. These include the following.

**(1)** There are many plausible contributors to the obesity epidemic beyond restaurant food. My colleagues and I recently reviewed 10 of them in a widely

discussed paper (Keith et al., 2006). Our thoughts have been echoed by other obesity researchers publishing in respected peer-reviewed journals (e.g., Astrup et al., 2006; Bray & Champagne, 2005; Eisenmann, 2006; Heindel, 2003; Jacobson et al., 2007; Newbold et al., 2007) and there are additional putative contributors beyond the 10 we mentioned (c.f., Pasarica & Dhurandhar, 2007).

**(2)** The evidence directly in favor of the unique role of restaurants as contributing to the obesity epidemic, is strictly from observational research, and more importantly, equivocal. The data cited in support of the unique role of restaurants rarely, if ever, stem from RCTs, and generally do not even include a consistent body of observational epidemiologic studies. I acknowledge that there are multiple epidemiologic studies showing a positive ('direct') association (not necessarily causation) between fast food and/or general restaurant consumption and overweight, obesity, or BMI (BMI denotes body mass index, a measure of relative weight [kg/m$^2$] used as a proxy for adiposity). Some of these studies were cited by the City, Dr. Frieden, and the amicus and there are others still that support a positive or direct association that they did not cite (e.g., Mehta & Chang, 2008; Schroder et al., 2007). Yet, there are also multiple studies that do not support this association (e.g., French et al 2001; Jeffery et al, 1998; Sanigorski et al., 2007; Sturm & Datar, 2005). The findings of French et al. (2001) are especially interesting. In a sample of 4,746 adolescents, "*Overweight status was not significantly associated with FFFRU [frequency of fast food restaurant use] among males or females. Interestingly, BMI was significantly lower among males who reported using fast food restaurants three or more times per week, compared with those reporting less frequent fast food restaurant use. BMI was*

*not significantly associated with FFFRU among females*" (French et al., 2001, p. 1828). That is, in this large sample, not only was BMI not positively correlated with frequency of fast food restaurant use, but it was negatively correlated in males.

**(3)** When correlations have been observed between fast food use and overweight, obesity, or BMI, such correlations are plausibly due to confounding. Fast food restaurants tend to be more prevalent in the poorest and most deprived neighborhoods (Block et al., 2004; Cummins et al., 2005; Reidpath et al., 2002) and poverty and deprivation have been associated with obesity (Phipps et al., 2006). Moreover, fast food restaurant use has been associated with having soda and chips in the home (Boutelle et al., 2007), more TV and video viewing (Taveras et al., 2006), and lower income (French et al., 2000), all factors that have been proposed or shown to be associated with obesity. Thus, these factors may be confounding the association (if any) between fast food consumption and obesity. The plausibility of confounding by these factors is increased by the fact that at least one study found fast food patronage/consumption to be more strongly associated with BMI or BMI gain than is other restaurant consumption (Duffey et al., 2007) and yet other restaurants have not been shown to have consistently more nutritionally sound food (Saelens et al., 2007) and may even provide higher calorie meals to many patrons (Yamamoto et al., 2006). This suggests the hypothesis that fast food patronage is correlated with BMI or BMI change more so than is other restaurant patronage because fast food patronage is a marker for lower socioeconomic status and/or other confounding factors.

Given the forgoing points, I concur with Jeffery et al. (2006) who wrote *"Available data on fast food use and obesity are far from conclusive, however. For example, the direction of causation is unclear, i.e., the menus and prices at "fast food" restaurants may result from the demands of an increasingly obese population rather than being a direct cause of obesity. It is also possible that a third variable, such as demographics and lifestyle characteristics (e.g. an aging population with smaller families and a higher percent of two income families), may cause both phenomena."*

Despite the reasons for skepticism about the truth of Proposition F5 I have offered above, I nevertheless believe that Propositions F1 to F3 are true, I accept proposition F4, and Proposition F5 is plausible. In turn, on this basis, I believe it is reasonable to conjecture that providing calorie information at the point of purchase in restaurants (especially fast food restaurants) might be beneficial in reducing obesity levels.

However, I believe it is equally reasonable to conjecture that providing calorie information at the point of purchase in restaurants (including fast food restaurants) would have no beneficial effect on reducing obesity levels. This is because providing such calorie information may have little to no effect on reducing caloric intake in the restaurants affected by R81.50 (Yamamoto et al., 2006), patrons may compensate for any reduced caloric intake that does occur by increasing caloric intake or decreasing caloric expenditures in other settings (Foltin et al., 1992; King et al., 2007; Whybrow et al., 2007), obesity-predisposed patrons may choose to frequent other restaurants not posting caloric information where overeating may be normative as they have been shown to do elsewhere (Stunkard & Mazer, 1978), or any effect of providing calorie information may be 'washed out' or overridden by the many other factors influencing body weight (Bray & Champagne, 2005). Moreover, in a survey of several hundred

persons, "If it were available, 44% to 57% of the combined sample self-reported that they would not likely use restaurant food caloric information" (Krukowski et al., 2006). Along these same lines, Cawley (2007, pp 38-39), commenting on the potential utility of providing public information such as calorie content as a means of reducing obesity wrote: "Because processing the information needed to make a large number of comparisons can be costly, people have been found to ignore information; for example, on health insurance plan options...suggesting that at some level of complexity consumers refuse to process information. These findings suggest that the market failure caused by lack of information regarding the calorie content of foods may not be resolved by simply providing more information..." Of course, this is all just conjecture, but it is well-reasoned conjecture based on the scientific literature. Given that one can reasonably conjecture in either direction, it is critical to turn to an assessment of the direct evidence (if any) that does or does not support the proposition that R81.50 will be effective in reducing obesity levels.

**G. Is there Competent and Reliable Evidence that (A) Providing Restaurant Patrons with Calorie Information On Menu Items in Any Manner Will Reduce Individual or Population Levels of Obesity; and (B) That Doing So in the Manner Mandated by R81.50 Will Reduce Obesity More Effectively Than Will Providing Such Information in Some Other Manner?**

This question, which is key to assessing the evidentiary basis for Regulation 81.50, must be broken down into a number of components to properly analyze the relevant studies.

First, one must distinguish between short and long-term effects[4]. In the obesity field, it is well-established that it is far easier to influence weight for a short period of time than for a long one. We have substantial data showing that weight control interventions that successfully alter behavior over the short term – days, weeks, or even months -- prove to demonstrate little to no efficacy over years (Hill et al., 2005). This may be related to 'set points' around which people maintain their weight, and the strong physiological signals that influence 'energy compensation' (Keesey, 1988). Studies show that altering caloric consumption or expenditure in one context can trigger compensatory consumption or expenditure in other contexts with no net change in weight (Foltin et al., 1992; King et al., 2007; Whybrow et al., 2007).

Second, it is important to distinguish between **(a)** the effects of providing caloric information in any format versus providing no caloric information at all; and **(b)** the effects of providing caloric information in formats that would be compliant with R81.50 versus some other format.

Third, as my colleagues and I have recently discussed elsewhere (Faith et al., 2007), it is important to not conflate outcome variables. Such outcomes include: **(1)** the patron's stated or actual evaluation of the desirability of high-calorie food; **(2)** the caloric content of the meal the patron actually orders and eats in naturalistic setting; and **(3)** the weight and obesity prevalence of the restaurant's patrons, which depend on their calorie consumption and expenditure from all sources throughout multiple days.

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[4] A standard, promulgated by the Federal Trade Commission, is to consider 'long-term' to be at least 2 years in the field of weight loss and obesity. See: http://www.ftc.gov/os/1997/12/d9261.htm. FTC states that when claims about evidence of long-term effects are made "said evidence shall, at a minimum, be based upon the experience of participants who were followed for a period of at least two years...".

Studies which measure an individual's *stated* (or real) intentions with respect to food purchase or consumption after receiving certain information are not necessarily indicative of what the individual will actually purchase and consume, especially over the long term and in natural settings. This can be because subjects do not actually know how they will behave in the future, because intentions are imperfect effectors of behavior, or because subjects know which answers experimenters want to hear and dutifully provide those verbal responses regardless of true intents or behaviors (Harnack et al., 2004). Studies which measure actual consumer purchase or consumption behavior are better, but are sill not as informative as studies which follow weight or obesity prevalence, since reduced consumption of calories in one setting may be counterbalanced by increased consumption or reduced expenditure in other settings as my colleagues and I have noted elsewhere (c.f., Faith et al., 2007).

For ease of exposition, using the distinctions elucidated above, I break the question posed in this section into 16 specific related questions in Tables 1 and 2 on the following pages and therein summarize the evidence regarding each.

| Table 1. | Effects of Providing Caloric Information in Any Format vs Not at All | | | Effects of Providing Caloric Information in the Format Required by R81.50 vs Some Other Format | | |
|---|---|---|---|---|---|---|
| | Nearly Relevant Studies | Relevant Studies | Level of Evidence[5] | Nearly Relevant Studies | Relevant Studies | Level of Evidence |
| **Short-Term Effects** | | | | | | |
| Stated intentions, attitudes, or preferences | Allen et al. (2007)[6]<br>Burton & Creyer (2004)[7]<br>Conklin et al. (2005)[8]<br>Kozup et al. (2003)[9]<br>Yamamoto et al., (2005)[10] | Burton et al. (2006) | **B** | None | Possibly[11] Burton et al. (2006) | **< D[12]** |
| Actual Purchases and/or Consumption | Dubbert et al. (1984)[13]<br>Kral et al. (2002)[14]<br>Sproul et al. (2003)[15]<br>Aaron et al. (1995)[16]<br>Mayer et al. (1987)[17]<br>Zifferblatt et al. (1980)[18]<br>Quesen (unpublished)[19]<br>DOHMH (2007)[20] | Milich et al. (1976)[21] | **< D[22]** | None | None | **< D*[23]** |
| Total Daily Consumption | None | None | **< D*** | None | None | **< D*** |
| Weight or Body Fat | None | None | **< D*** | None | None | **< D*** |

[5] Evidence levels are defined in the table reproduced after the following page.
[6] Not a randomized study; instead a pre-post quasi-experiment. Also not directly relevant because the independent variable is multiple bits of nutrition information, not just calorie content (see discussion below).
[7] Not directly relevant because the independent variable is multiple bits of nutrition information, not just calorie content (see discussion below).
[8] Not a study of calories alone. Also not a randomized experiment. No control group.
[9] Not directly relevant because the independent variable is multiple bits of nutrition information, not just calorie content (see discussion below).
[10] Not directly relevant because the independent variable is calorie plus fat content, not just calorie content.
[11] I write 'possibly' because it is unclear whether the comparison used is one that would be critical. The comparison entailed calorie information alone compared to calorie information with additional nutrition information and evaluated whether there was any interaction of the provided nutrition information with 'context information' involving provision of recommended daily values (see discussion below).
[12] That is, the one possibly relevant study does not show consistent evidence supporting superiority of one method of providing calorie information relative to another.
[13] Only the foods with the lowest calories were labeled.
[14] Study was in a laboratory setting, not a restaurant setting. Results showed no effect of provision of caloric density (not total kcal) information on energy intake.
[15] Not directly relevant because the independent variable is multiple bits of nutrition information, not just calorie content.
[16] Not directly relevant because the independent variable is calorie and fat content, not just calorie content.
[17] Not a randomized design. Intervention included more than providing caloric information. There was no significant reduction in calories purchased during intervention period.
[18] Not a randomized experiment. Intervention included more than providing caloric information.
[19] Not a randomized design. Information provided at point of purchase included more than calories. Authors concluded "the mean difference was only 2.06 calories per person. Therefore, there was not a substantial reduction in calories as a result of the intervention. The hypothesis of no difference cannot be rejected..."
[20] Not a completed study. No randomization or even experimental manipulation of independent variable. No inferential statistical analysis reported (see discussion below).
[21] This is not a randomized study, calorie information may have been confounded with price changes, and the statistical analysis was quite questionable.
[22] Dubbert et al "indicated [calorie] labeling did not change the total caloric content of meals, " Sproul et al. reported "Analysis of variance detected no significant differences in sales between baseline and the two intervention periods," and Aaron et al found providing fat and calorie information ***actually increased*** calorie and fat intake.
[23] I use the notation "<D*" to denote situations where I have been unable to identify even a 'Blue Ribbon' panel endorsing the belief in the proposition, but should such a panel report be provided to or identified by me, I would, depending on the content, potentially change the rating from <D to D.

| Table 2. | Effects of Providing Caloric Information in Any Format vs Not at All | | | Effects of Providing Caloric Information in the Format Required by R81.50 vs Some Other Format | | |
|---|---|---|---|---|---|---|
| | Nearly Relevant Studies | Relevant Studies | Level of Evidence[24] | Nearly Relevant Studies | Relevant Studies | Level of Evidence |
| **Long-Term Effects** | | | | | | |
| Stated intentions, attitudes, or preferences | None | None | **< D*** | None | None | **< D*** |
| Actual Purchases and/or Consumption | None | None | **< D*** | None | None | **< D*** |
| Total Daily Consumption | None | None | **< D*** | None | None | **< D*** |
| Weight or Body Fat | None | None | **< D*** | None | None | **< D*** |

[24] Evidence levels are defined in the table reproduced on the following page.

| Evidence category | Sources of evidence | Definition |
|---|---|---|
| A | Randomized controlled trials (rich body of data) | Evidence is from endpoints of well-designed RCTs (or trials that depart only minimally from randomization) that provide a consistent pattern of findings in the population for which the recommendation is made. Category A therefore requires substantial numbers of studies involving substantial numbers of participants. |
| B | Randomized controlled trials (limited body of data) | Evidence is from endpoints of intervention studies that include only a limited number of RCTs, post hoc or subgroup analysis of RCTs, or meta-analysis of RCTs. In general, Category B pertains when few randomized trials exist, they are small in size, and the trial results are somewhat inconsistent, or the trials were undertaken in a population that differs from the target population of the recommendation. |
| C | Nonrandomized trials observational studies | Evidence is from outcomes of uncontrolled or nonrandomized trials or from observational studies |
| D | Panel consensus judgment | Expert judgment is based on the panel's synthesis of evidence from experimental research described in the literature and/or derived from the consensus of panel members based on clinical experience or knowledge that does not meet the above-listed criteria. This category is used only in cases where the provision of some guidance was deemed valuable but an adequately compelling clinical literature addressing the subject of the recommendation was deemed insufficient to justify placement in one of the other categories (A through C). |

*Note.* See http://www.nhlbi.nih.gov/guidelines/obesity/ob_gdlns.htm. From *NHLBI Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults—the Evidence Report*, by the National Heart, Lung, and Blood Institute, 1998, Bethesda, MD: Author. Reprinted with permission. RCT = randomized clinical trial.

In Tables 1 and 2, I have included every paper that the City, Dr. Frieden, and the amicus cited as supportive of the likely[25] efficacy of R81.50. In addition, I include other papers that I was able to identify. I have searched fairly extensively and have been unable to identify any additional relevant studies.

For example, the primary support relied on by the City and by Dr. Frieden as evidence that R81.50 will 'likely' have its intended effect on obesity are three papers by Burton and colleagues (Burton et al., 2006; Kozup et al., 2003; Burton & Creyer, 2004) and their own observational study (DOHMH, 2007). The studies by Burton and colleagues report on the impact of providing nutritional information on consumers' attitudes to restaurant meals and/or self-reported willingness or intentions to purchase foods and have important methodological limitations. Perhaps most critically, they focus on the effects of providing a different sort of nutritional information than that required by Regulation 81-50, and cannot provide evidence regarding the effects (or lack thereof) of the specific practices that would be mandated by R81.50.

The 2003 and 2004 papers report several experiments on the effect of providing nutritional information to consumers regarding a hypothetical restaurant meal such as lasagna or pot roast. The authors report a positive impact of providing information on the fat, saturated fat, calories-from-fat and cholesterol levels of the meals. The calorie-only information required by R81.50 was not tested. The 2006 study did give some

[25] I note that both Dr. Frieden and the amicus use the term 'likely' and are quite clear in there language that R81.50 is only ***likely*** to be effective. They never state that there is evidence showing that it ***will*** be effective.

subjects calorie-only information and others the fat, cholesterol, etc information, and found no advantage to the calorie-only option. Each of the studies tested the impact of menu information by having subjects presented with a hypothetical menu complete a questionnaire on their subjective impressions after reading it, e.g., "How likely would you be to purchase this item given the information shown on the menu?" No meals were served, and actual purchases and consumption were not measured. Subjects' overall consumption and weight were not measured. Each study only measured the impact of information on a single occasion. Effects over days or weeks, and certainly impact on long-term weight management, was not tested. The studies focus on meals served at what the authors describe as "dinner house restaurants", Menu boards were not used to provide the nutritional information. Subject samples were not representative of the NYC population or the population of fast food diners, and response rates tended to be low suggesting concerns about generalizability of findings. The findings do not show that providing nutritional information at the type of restaurants generally subject to R81.50, and in the specific manner R81.50 requires, will be effective at all or more effective than alternative modes of providing nutritional information.

***The DOHMH's Own Study.***

The City's own study (DOHMH, 2007) has many very substantial limitations, falls markedly below accepted standards for research in the scientific community, is interpreted in a scientifically unjustifiable manner, and does not offer competent and reliable evidence regarding any effects or R81.50.

In brief, DOHMH conducted a survey in a sample (which they describe as representative) of major restaurant chains in NYC during March through June 2007. They collected information from 11,835 diners at a random sample of 275 restaurants. Field

teams visited the restaurants and recorded whether and by which means calorie information on menu items was provided. Customers departing the restaurants were asked to supply their restaurant receipt and to answer a number of questions. DOHMH reports that "For the 7,308 patrons who purchased one or more items, for themselves only, at one of 11 major restaurant chains surveyed on a weekday between 12 noon and 2 PM – a total of 168 locations across the five boroughs – the average calorie purchase was 824 calories ***(preliminary data)*** [emphasis added]."

The study was designed to compare (1) purchases at chain restaurants before implementation of R81.50 with (2) purchases at the same restaurants after implementation, in order to evaluate the association (it cannot evaluate a causal effect) of implementation of the regulation with purchasing behavior. The second half of the study has not been conducted since R81.50 has not gone into effect. The study was not designed to draw inferences from just the pre-implementation portion completed to date.

The DOHMH states that it "was able to examine the ***impact*** [emphasis added]of point of purchase calorie information at Subway sites, New York City's second largest chain with 315 locations....Subway posted nutritional information for some of its products on a sticker placed on a display case near the cash register – a manner far less prominent than that mandated by §81.50. ... Patrons who saw calorie information purchased items containing 48 fewer calories than those who did not see this information. Furthermore, patrons who said calorie information had affected their selection were correct – they chose items with 92 fewer calories. ... Based on the best estimates, if the reduction in calories in covered FSEs were similar to what occurred at Subway, over the next five years at least 150,000 fewer New Yorkers would be obese,

resulting, among many other health benefits, in at least 30,000 fewer cases of diabetes, and possibly many more than that."

There are many serious limitations[26] and flaws[27] in the DOHMH study. These limitations and flaws make it of little to no value in estimating the effects of R81.50 and inappropriate for drawing the conclusions that the DOHMH drew in the quotation above. These include, but are not necessarily limited to the following.

***Limitations.***

- Although the restaurants may be a random sample of restaurants in NYC, this does not insure that the sample of participants is or can be expected to be a representative sample of NYC residents, NYC restaurant diners, or any other population of humans.
- The study involves no random assignment or even experimenter manipulation of the independent variable (provision of calorie information in specific forms) and therefore justifies no statements about causation.
- The study does not appear to permit an evaluation of even the association of information in the specific form required in R81.50 versus no information or information provided in a different manner.
- The study provides no information on long-term effects on weight or even on total energy intake and expenditure across the single day in which any particular patron was assessed and therefore cannot take into account

[26] By limitations, I mean factors inherent in the study itself that limit its value.

[27] By flaws, I mean avoidable errors made by the study investigators and/or DOHMH that were not essential to the study itself.

compensation in energy balance that is known to occur in many situations (e.g., Foltin et al., 1992).

***Flaws.***

- No inferential statistics are presented, so we are unable to discern if any of the associations reported are statistically significant.
- No effort was made to control for potential confounding factors such as subjects' age, race, sex, education level, income, interest in nutrition, body mass index, etc. To do so would be standard practice in epidemiological research.
- The DOHMH inappropriately uses language implying causation when the observational design can support no causal conclusions.
- The DOHMH suggests that the data it collected show that "point of purchase" posting of calorie information on a sticker near the cash register at Subway restaurants proved a more effective way of drawing the information to the attention of patrons, and influencing them to order lower-calorie items, than alternative methods. DOHMH contrasts that method with other methods such as providing calorie information in brochures, on placemats or on food wrappers, which it criticizes as "woefully inadequate". The actual DOHMH study records, however, which document how calorie information was provided in the Subway restaurants studied, report that the calorie information was displayed in a variety of ways similar to those DOHMH characterizes as inadequate. In a number of Subway restaurants, the

information was displayed on cups and napkins; in others, it was in pamphlets; in others, on posters; in just a few it was on a display case decal.[28]

- Little or no information is presented as to how the projection[29] offered above was conducted. Such projections require many assumptions and complex statistical modeling. Because the basis of the projection is not made clear, as would be accepted scientific practice, it is difficult to evaluate the reasonableness of the projections. Nevertheless, I can offer several comments on what appear to be components of DOHMH's methodology.
    - First, it appears that DOHMH used an estimated 'differential' of 48 or 92 kilocalories (kcal) per customer per meal purchased as the effect of R81.50. If so, this is unjustified because: (a) It is based on only patrons at Subway restaurants who likely differ from patrons at other restaurants (c.f., see Chandon & Wansink 2007); (b) It is based only on those that report having seen the nutrition information who likely differ from individuals who did not notice (or report noticing) the information; (c) the 92 kcal is based only on those that report having had their selections influenced by viewing the nutrition information who almost assuredly differ from individuals who did not report this and is tantamount to selecting for the very outcome one wishes to

[28] "Observation Logs" for Subway, various dates. DOHMH (2007).

[29] By projection, I refer to this statement "Based on the best estimates, if the reduction in calories in covered FSEs were similar to what occurred at Subway, over the next five years at least 150,000 fewer New Yorkers would be obese, resulting, among many other health benefits, in at least 30,000 fewer cases of diabetes, and possibly many more than that."

study. Thus, if the 92 kcal value was used as the estimated mean effect, this is assuredly invalid.

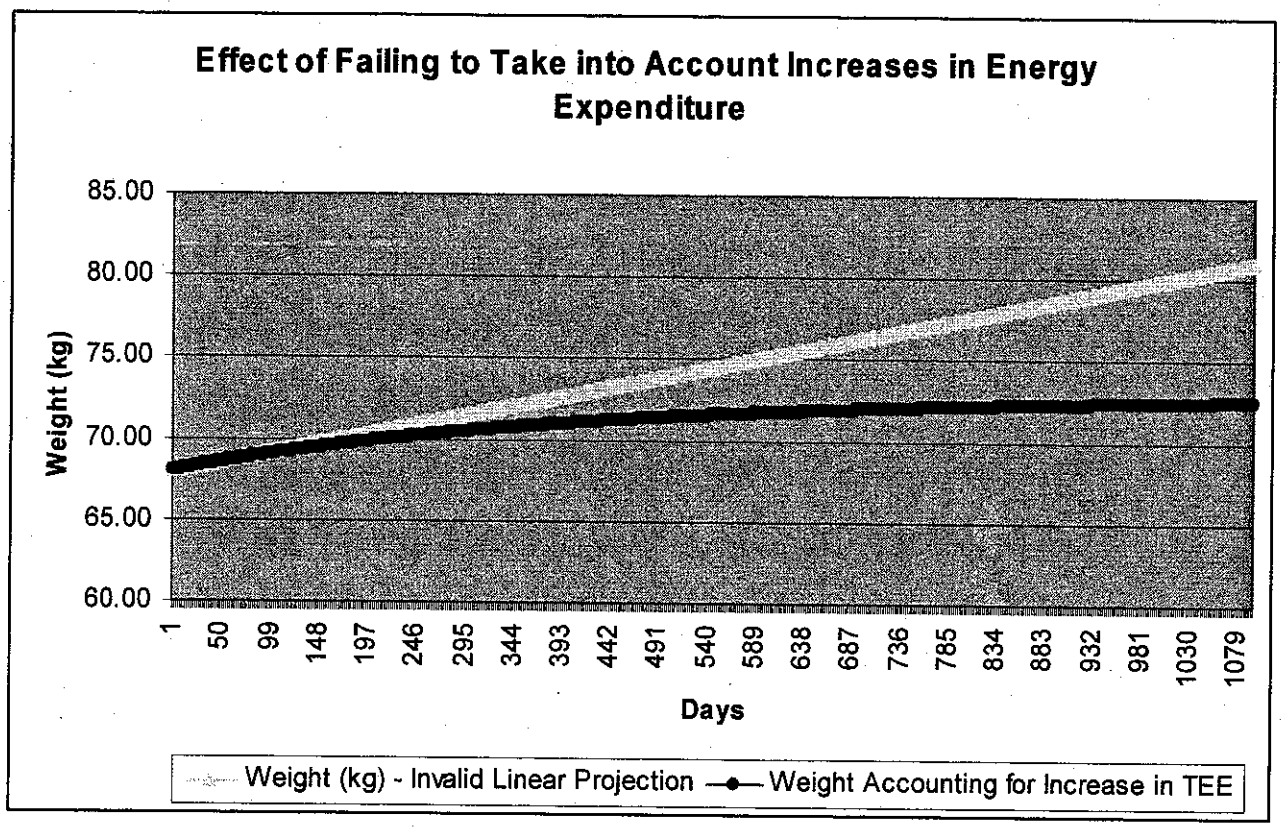
- Second, regardless of which specific kcal estimate DOHMH used as the projected effect of R81.50, if DOHMH only used a single point estimate (as opposed to evaluating the full multivariate distribution of the calorie differential and other variables to be used in the projection), then the projected estimate is almost assuredly invalid, because it assumes a constant effect on all persons. In contrast, not only might any effects of R81.50 on calorie intake differ as a function of subject characteristics (e.g., age, sex, education, BMI), but the effects of any differential in calorie intake on obesity and other health outcomes is likely to differ by participant characteristics. Consider a contrived hypothetical illustration to simply explain this point. Suppose that 50% of patrons were females under age 25 with BMIs under 20 who, being very concerned about their weight sought out and attended to the nutrition information and reduced their purchases by 2x92 = 184 kcal. Suppose further that the other 50% of patrons were males over age 45 with BMIs over 25 who, were uninterested in this information and reduced their purchases by 0x92 = 0 kcal. Then, on average, there was a 92 kcal differential. Yet, all of the differential was limited to a group at extraordinarily low risk of obesity and diabetes, whereas there is no differential among a group at substantial risk of obesity and diabetes. Under such circumstances,

we would expect virtually no effect of projected a 92 kcal effect R81.50 on obesity or diabetes.

- Based on other statements of the DOHMH[30], it appears that the DOHMH is assuming that: (a) it takes roughly 3,500 kcal increment above energy utilized for an adult human to add a pound of body weight; and (b) As a person increases in mass, their energy expenditure remains constant. Assumption (a), though a rough approximation, is probably a reasonable one (Pietrobelli et al., 2002). However, assumption (b) is patently invalid and leads to a marked overestimation of effects of calorie differentials. This mistake has been pointed out numerous times in the scientific literature on obesity (Weinsier et al., 1993; Mattes, in press), but remains unfortunately common. The impact of the error is illustrated in the figure below, which I have based on a figure from Mattes (in press). In the figure, I calculate expected weight gain of a hypothetical 45 year old, 1.7 meter, 68 kg woman who had been in energy balance but, on day 1 of the graph, increases her intake 100 kcal/day. The upper line is the expected weight gain using the naïve linear calculations that DOHMH use. The lower line is the expected weight gain allowing for increases in total energy expenditure (TEE) using a formula from

[30] E.g., "In the second survey of more than 9,000 adults, mean energy intake on days when fast food was consumed was 206 calories higher than on other days. This increase in calories would result in a three pound weight gain each year if a consumer were to eat fast food only once each week."

Vinken et al. (1999). As can be seen, the error induced by using a linear extrapolation can be quite substantial.




***Summary on Evidence (or Lack Thereof) Regarding the Expected Effects (or Lack Thereof) of R81.50.***

In brief, there is reasonably persuasive evidence (level B) that providing caloric information (vs no calorie information) will change stated purchase intentions in the short term. Beyond that, there is no body of data that showing that implementation of R81.50 would affect actual behavior or weight either in the short-term or long-term nor is there any body of evidence that the specific manner in which the R81.50 would require provision of caloric information would lead to better results in the short-term or long-term than any other method. **Thus, I conclude that there is not competent and reliable**

**evidence that providing restaurant patrons with calorie information on menu items will reduce individual or population levels of obesity. Nor is there evidence that the method of providing caloric information mandated by R81.50 will reduce levels of obesity more than the methods currently used by the affected restaurants to provide this information.** This does not imply that there is competent and reliable evidence that providing restaurant patrons with calorie information on menu items will not reduce individual or population levels of obesity. It simply implies that there is insufficient evidence to draw a conclusion.

## H. Are There Reasons to Conjecture That There Might Be Unintended Deleterious Effects of the City's Proposed Rule?

Intuition might suggest that, at worst, R81.50 could do no harm to the public health. Dr. Frieden seems to share this intuition when he writes "And even in the unlikely event the calorie labeling regulation had little or no impact on consumer food choices, it is likely to increase the number of lower-calorie, and reduce the number of higher-calorie offerings these facilities provide. And even if the regulation neither changed consumer choices nor FSE offerings, there would be a salutary effect on increasing consumer awareness of calorie content." Nevertheless, it is important to note that the history of science, medicine, and psychology tells us that intuitions, even those of well-trained experts, are often wrong especially on issues involving human behavior (Dawes et al., 1989).

To take just a few examples, few people would argue that we should not try to prevent post-traumatic stress disorder (PTSD) among NYC rescue workers, families, and direct victims after the tragedy of September 11, 2001, teen suicide, adolescent eating disorders, and childhood sexual abuse. And yet, in each of these areas, evidence has

emerged that the well-intentioned and intuitively sound efforts of professionals may have some times done more harm than good (Carter et al., 1997; Callahan, 1996; Taal & Edelaar, 1997; McNally, 2003).

Closer to the specific issue herein and highlighted in a recent Associated Press report entitled "Nutrition education ineffective[31]," is a recent CDC-sponsored study (Centers for Disease Control and Prevention, 2006). In 2004-05 a program was implemented to "1) increase student access to fresh fruit and vegetables, 2) increase the degree of student preference for fruit and vegetables, and 3) increase fruit and vegetable consumption." Participating schools distributed free fresh fruit and vegetables to school children accompanied by nutrition education activities. Results showed a *decreased* preference for fruit and vegetables, belief that they could eat more vegetables, and willingness to try new fruit and vegetables among 5th-grade students. In other words, for these children, the seemingly innocuous intervention seemed to make things worse.

What harms (if any) might result from implementation of R81.50? That is difficult to predict. An interesting study by Boon et al. (2002) points to one possibility. Boon et al. found that when restrained eaters[32], who constitute roughly 50% of the population according to the definition Boon et al. used, were both distracted by being asked to perform a cognitive task and also presented with information that the food they were eating was high in calories, they were ***more*** likely to overeat. To the extent that many NY diners consume food from restaurants while in a state of distraction or performing

[31] Nutrition education ineffective By Martha Mendoza, Associated Press. See: http://www.usatoday.com/news/health/2007-07-04-fightingfat_N.htm

[32] Restrained eaters are those that habitually tend to eat less than they want despite the availability of food.

distracting tasks, we might hypothesize that the belief that the food is especially high in calories would trigger disinhibited increased consumption.

The study by Aaron et al. (1995) cited in Table 1 is especially germane. Aaron et al. "examined the influence of nutrition information on nutrient intakes at lunch in a college cafeteria...Experimental subjects (EXP, n=65) ate all mid-day meals in a student cafeteria over two weeks with identical meal cycles. Information on the energy and fat content of food items was displayed in the cafeteria in week two. ...Unexpectedly, EXP subjects had significantly increased total energy, grams fat, grams carbohydrate and decreased grams protein and % energy from protein in week two vs. week one." Thus, Aaron et al's results were opposite to the intention of providing nutrient and calorie information and *worsened* subjects' diets.

With respect to deleterious effects, other conceivabilities include (but are not limited to) creation of greater interest in foods seen as 'decadent' or 'forbidden' (Jansen et al., 2007), inadvertently encouraging patrons to consume lower calorie, but arguably less healthy choices (e.g., a food product that is largely refined sugar versus one that is largely protein), and inadvertently encouraging patrons to consume lower calorie foods that subsequently lead to greater total caloric intake because of poor satiating efficiency of the smaller calorie loads (Booth, 1988) and/or because of cognitively mediated reactivity to the label-induced perception of what one has eaten (Caputo & Mattes, 1993).

The foregoing does not by any means prove that there will be deleterious outcomes if R81.50 is implemented, but it shows that the conjecture is reasonable. Moreover, the belief that providing mandatory nutritional information for foods served in restaurants is plausibly nil and the conjecture that it could even be harmful is one

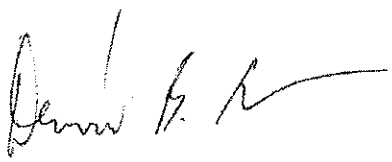
voiced by groups as mainstream as scientists from the United States Department of Agriculture's Economic Research Service (Kuchler et al., 2005).

## I. Summary.

In summation I believe that **(1)** the body of scientific evidence clearly supports the merit of concerns about obesity and suggests that as a society we should be seeking ways to reduce obesity and/or the deleterious effects it causes. **(2)** Though there are some limitations to the evidence available, on balance, the evidence can support the *conjecture* that provision of information about the calorie content of foods at the point of purchase in restaurants might be beneficial in reducing obesity levels. That being said, the evidence can equally support the conjecture that such action would be ineffective and possibly even deleterious; and **(3)** There is not competent and reliable evidence that providing restaurant patrons with calorie information on menu items will reduce individual or population levels of obesity. Nor is there evidence that the method of providing caloric information mandated by R81.50 will reduce levels of obesity more than the methods currently used by many of the affected restaurants to provide this information.

I declare under penalty of perjury pursuant to 28 U.S.C. § 1742 that the foregoing is true and correct.

Executed on January 29, 2008



David B. Allison, Ph.D.

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